CHAPTER 7
Stance and Gait

KEY TEACHING POINTS

- Observation of the patient's gait helps diagnose important neurologic and musculoskeletal problems and allows clinicians to predict the patient's risk of falls.
- Gait abnormalities may be symmetric or asymmetric. Pain, immobile joints, and muscle weakness cause *asymmetric* gaits. Rigidity, proprioceptive disorders, cerebellar diseases, and problems with central control all cause *symmetric* gaits. Spasticity may cause asymmetric gait abnormalities (i.e., hemiplegia) or symmetric ones (i.e., paraplegia).
- Simple observation may result in prompt diagnosis. Examples include the *lateral lurch* of hip disease, the *backward lean* of gluteus maximus weakness, the *Trendelenburg gait* of gluteus medius weakness (often after hip replacement), the *steppage gait with foot slap* of foot drop, the *leg circumduction* of hemiplegia, and the *shuffling steps with narrow base and flexed posture* of Parkinson disease.
- Gait abnormalities are prominent in Lewy body dementia and vascular dementia but are uncommon in Alzheimer dementia until late in its course.
- The timed-up-and-go test, stops-talking-when-walking test, and observation of the patient's ability to stand with feet together for 10 seconds all accurately assess the elderly patient's risk of falls.

I. INTRODUCTION

Observation of gait not only uncovers important neurologic and musculoskeletal problems (e.g., Parkinson disease, hemiparesis, spinal stenosis, hip disease), but it also provides clues to the patient's emotions, overall function, and even prognosis. For example, the speed of an elderly person's gait accurately predicts falls, future disability, and risk of institutionalization.[1-5] In patients with congestive heart failure, gait speed predicts cardiac index, future hospitalization, and mortality, as well as the ejection fraction and better than the treadmill test.[6,7] Even depressed patients have a characteristic gait, marked by an abnormally short stride and weak lift-off of the heel.[8]

The phases of the normal gait are depicted in Fig. 7.1.

II. ETIOLOGY OF GAIT DISORDERS

Among patients presenting to neurologists, the most common causes of gait disorder are stroke and Parkinson disease, followed by frontal gait disorder, myelopathy (e.g., cervical spondylosis, B12 deficiency), peripheral neuropathy, and cerebellar disease.[9,10] Among patients presenting to general clinicians, most gait abnormalities

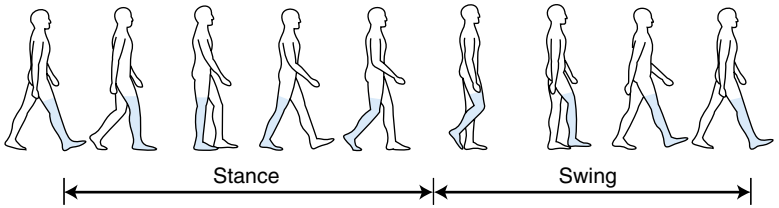


FIG. 7.1 NORMAL GAIT. This figure illustrates the phases of normal gait, focusing on the right leg (*colored gray*). Normal gait consists of the *stance phase* (the period during which the leg bears weight) and *swing phase* (the period during which the leg advances and does not bear weight). The stance and swing make up the *stride*, which is the interval from the time one heel strikes the ground to when it again strikes the ground. During the normal stance phase, it is the *extensor* muscles that contract—the gluteus maximus in early stance, the quadriceps in mid-stance, and the plantar flexors (soleus and gastrocnemius) in terminal stance, pushing off the heel. The healthy swing, in contrast, requires contraction of the *flexor* muscles, all of which are activated early in the swing phase—hip flexors (iliopsoas muscles), knee flexors (hamstring muscles), and ankle flexors (tibialis anterior and toe extensor muscles).[15,16] (Based upon reference 15.)

are caused by arthritis, followed by orthostatic hypotension, stroke, Parkinson disease, and intermittent claudication.[11]

III. TYPES OF GAIT DISORDERS AND THEIR SIGNIFICANCE

Disorders of gait reflect one of four possible problems: pain, immobile joints, muscle weakness, or abnormal limb control. Abnormal limb control, in turn, may result from spasticity, rigidity, diminished proprioception, cerebellar disease, or problems with cerebral control.

When analyzing a patient's gait, the most important initial question is whether the gait is symmetric or asymmetric. Pain, immobile joints, and muscle weakness are usually unilateral and thus cause *asymmetric* abnormalities of gait. Rigidity, proprioceptive disorders, cerebellar diseases, and problems with central control all cause *symmetric* abnormalities of the gait. Spasticity may cause *asymmetric* gait abnormalities (hemiplegia) or *symmetric* ones (paraplegia).

A. PAINFUL GAIT (ANTALGIC GAIT)

If bearing weight on a limb is painful, patients adopt an antalgic gait to minimize the pain. (*Antalgic* is from the Greek *an* and *algesis*, meaning "against pain.") All antalgic gaits are characterized by a short contralateral step, along with other characteristic features.

1. SHORT CONTRALATERAL STEP

After bearing weight on the affected leg, patients with pain quickly step onto the sound leg. The short contralateral step produces an uneven cadence, one identical to that produced by a rock in one shoe.

2. OTHER CHARACTERISTIC FEATURES

Depending on whether the pain is located in the foot, knee, or hip, each antalgic gait is distinctive, allowing diagnosis from a distance.

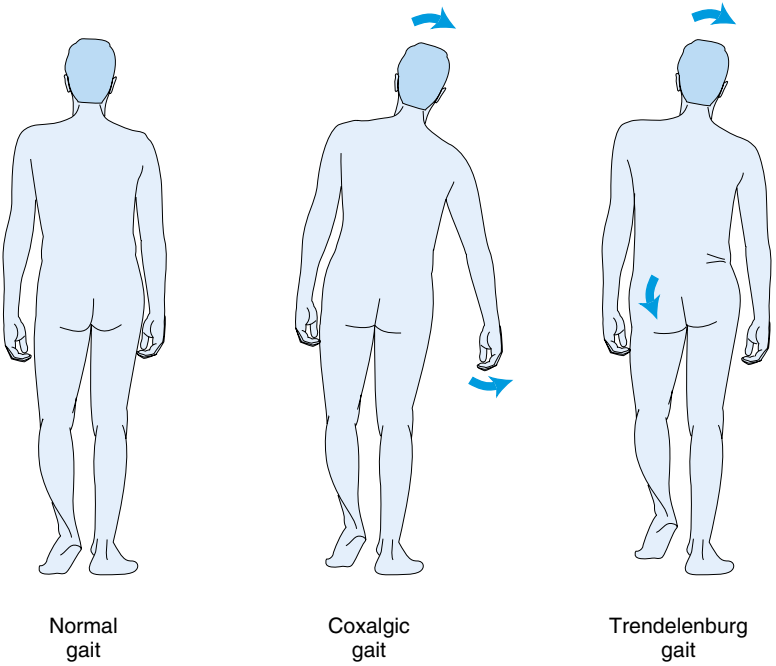


FIG. 7.2 COMPARISON OF COXALGIC AND TRENDELENBURG GAIT. In both abnormal gaits (middle and right figures), the trunk may lean over the abnormal leg during stance (*arrow*), but in patients with hip pain (coxalgic gait, middle figure), the trunk lean and accompanying ipsilateral arm movement (*arrow*) is more dramatic (lateral lurch), and the opposite pelvis does not fall excessively. In the Trendelenburg gait (from ineffective or weak hip abductors, right figure), the opposite pelvis falls excessively (*arrow*), and the conspicuous but opposing swings of the upper body and pelvis create the impression of a hinge between the sacral and lumbar spine. In these figures, the patient is bearing weight on the affected side (i.e., *right* hip pain [coxalgic gait] and ineffective *right* hip abductors [Trendelenburg gait]).

A. FOOT PAIN

In patients with foot pain, the foot contacts the ground abnormally. For example, patients may bear weight during stance on their heel only, forefoot only, or along the lateral edge of the foot.

B. KNEE PAIN

Patients with knee pain display a stiff knee that does not extend or flex fully during stride.[12]

C. HIP PAIN (COXALGIC GAIT)

Patients with hip pain limit the amount of hip extension during late stance (when the normal hip extends 20 degrees). Even so, the most characteristic feature of the coxalgic gait is the so-called lateral lurch: when bearing weight on the painful limb, there is an excessive asymmetric lateral shift of the patient's upper body toward the weight-bearing side, causing the trunk to lean and ipsilateral arm to abduct (Fig. 7.2).[13,14]

Lateral lurch reduces the pain of patients with hip disease because it minimizes the need to activate the ipsilateral hip abductor muscles. These muscles normally

support the upper body during swing of the other leg, but when activated can easily put 400 pounds of pressure on the femoral head, an intolerable force if there is hip disease. By leaning over the painful limb during stance, patients effectively balance their center of gravity over the painful limb and thus avoid activation of the hip abductors.

B. IMMOBILE JOINTS

Most clinicians do not consider immobile joints as a cause of abnormal gait, but the condition is well known to physiatrists. A common example is plantar flexion contracture, a complication that may occur after prolonged periods of plaster immobilization or confinement to bed. Affected patients may place their weight on the forefoot during initial stance (instead of the heel) or, during mid-stance, lift their heel too early or lean their trunk forward. During swing phase, the abnormally flexed foot has difficulty clearing the floor, leading the patient to drag the foot or develop an unusual movement to clear it, such as contralateral trunk lean or contralateral vaulting.[15,16]

The clinician can easily identify immobile joints as the cause of abnormal gait by testing the range of motion of hips, knees, and ankles of both legs.

C. WEAKNESS OF SPECIFIC MUSCLES

Three muscle groups, when weak, cause specific gait abnormalities: (1) hip extensor and abductor muscles (i.e., gluteus maximus and medius/minimus muscles), (2) knee extensors (quadriceps muscle), and (3) foot and toe dorsiflexors (tibialis anterior and toe extensor muscles). Gluteus maximus and quadriceps gaits were frequently observed historically as complications of poliomyelitis or diphtheria.

1. TRENDELENBURG GAIT AND SIGN (ABNORMAL GLUTEUS MEDIUS AND MINIMUS GAIT)

A. DEFINITION OF TRENDELENBURG GAIT (OR TRENDELENBURG'S SYMPTOM; FRIEDRICH TRENDELENBURG 1844–1924)

The Trendelenburg gait occurs when the gluteus medius and minimus do not function properly. These two muscles abduct the hip, an action that supports the opposite pelvis and prevents it from dropping excessively during the normal single-limb stance. During walking, a slight dip of the opposite pelvis is normal during the stance phase on one limb. An excessive drop of the opposite pelvis indicates an abnormal Trendelenburg gait. When the abnormality is bilateral, the pelvis waddles, reminiscent of a duck.

Like patients with the coxalgic gait (see previous section on Hip Pain/Coxalgic Gait), patients with Trendelenburg gait may lean their trunk over the abnormal leg during stance, but the lean lacks the dramatic lurch seen in coxalgic gait, and the opposing sways of the ipsilateral shoulder and opposite pelvis make it appear as if patients with Trendelenburg gait have a hinge between their sacral and lumbar spine (see Fig. 7.2).[14,17]

B. ETIOLOGY OF TRENDELENBURG GAIT

Causes include (1) neuromuscular weakness of the hip abductors and (2) hip disease. Although poliomyelitis and progressive muscular atrophy were important causes historically, this gait now occurs as a complication of hip arthroplasty using a lateral approach, which risks damage to the superior gluteal nerve or gluteus medius muscle.[18,19] Another common cause is congenital dislocation of the hip and coxa

vara (i.e., bent hip, a deformity in which the angle between the femoral neck and body is significantly decreased). In congenital hip dislocation and coxa vara, the abnormal upward displacement of the greater trochanter shortens the fibers of the gluteus medius, rendering them more horizontal than vertical and thus abolishing their role as abductors.

C. TRENDELENBURG SIGN

In 1895, before use of roentgenography, Friedrich Trendelenburg was the first to show that the waddling gait of patients with congenital dislocation of the hip was due to weak abductor function, not the upward movement of the femur during stance (which was what his contemporaries believed). He successfully argued this by inventing a simple test, now known as the Trendelenburg sign. In this test, the patient is asked to stand on one leg with the other hip flexed to 90 degrees (the clinician may help the patient balance by supporting the ipsilateral arm to align the ipsilateral shoulder over the hip being tested).[20] In patients with normal abductor strength, the contralateral buttock rises, but if the abductor muscles are weak, the contralateral buttock falls. (The buttock falls until the ipsilateral femur and pelvis come into contact.) It is important to remember that the side being tested is the one bearing the weight. Some deformities of the leg, such as severe genu varum, may cause a false-positive result.[21]

D. CLINICAL SIGNIFICANCE

In one study of patients clinically diagnosed with "trochanteric bursitis" (i.e., lateral hip pain and maximal tenderness over the greater trochanter),[22] the finding of both a positive Trendelenburg sign and gait on the symptomatic side accurately detected the MRI finding of a tear in the gluteus medius tendon (sensitivity = 73%, specificity = 77%, positive likelihood ratio [LR] = 3.2, negative LR not significant). This sign was superior to directly testing gluteus medius strength (by resisting the patient's active hip abduction or internal rotation, LRs not significant). The results of this study suggest that some patients with "trochanteric bursitis" actually have tendonitis or tears of the gluteus medius tendon, a discovery analogous to the historic realization that many patients with "subacromial bursitis" (in the shoulder) actually have disorders of the rotator cuff tendons.

In patients with a foot drop, the presence of ipsilateral hip abductor weakness argues that the foot drop is from lumbosacral radiculopathy and not peroneal nerve palsy (see Chapter 64). In one study of patients with foot drop from various causes, ipsilateral hip abductor weakness was a compelling sign of lumbosacral radiculopathy (positive LR = 24; see Chapter 64).[23] Although hip abductor weakness in this study was identified by manual resistance testing, the abnormality is often first suspected by observing a Trendelenburg gait.

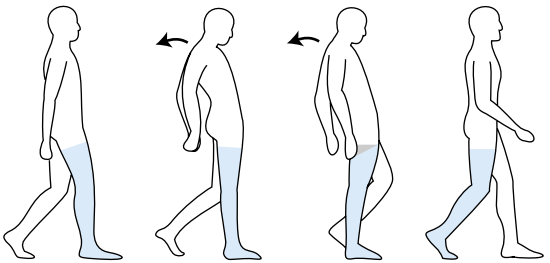
2. GLUTEUS MAXIMUS GAIT

If the hip extensors are weak, the patient develops a characteristic abnormal backward trunk lean during early stance, which places the patient's center of gravity behind the hip joint line and removes the need for the gluteus maximus muscle to contract (Fig. 7.3).

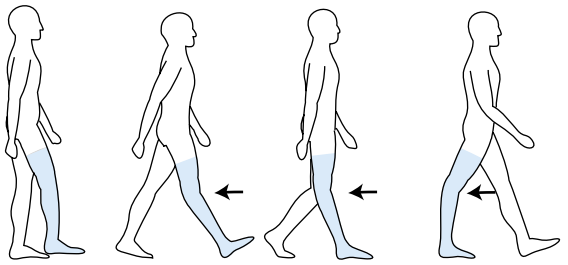
3. WEAK QUADRICEPS GAIT

If the knee extensors are weak, two different abnormalities of gait may appear. Some patients develop a characteristic hyperextension of the knee during stance (see Fig. 7.3). At first this seems paradoxical because the normal action of the quadriceps is knee extension, which should therefore be weak in these patients. However, the

Weak gluteus maximus gait



Weak quadriceps gait



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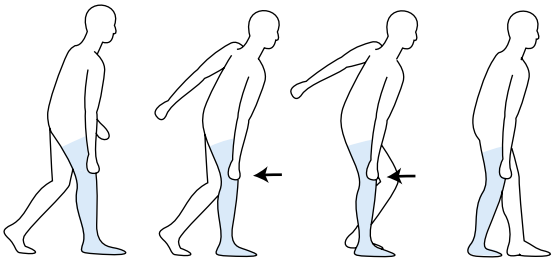


Footdrop gait

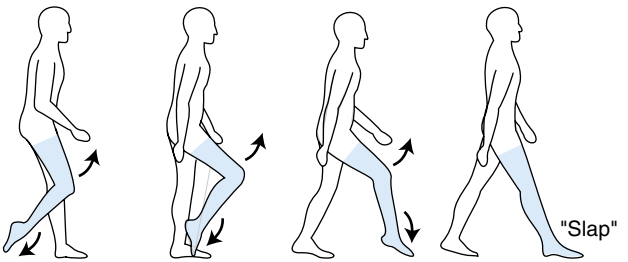


FIG. 7.3 CHARACTERISTIC GAITS OF WEAK MUSCLES. In each figure, the shading indicates the limb with the weak muscle and the black arrows indicate the diagnostic movements. Because both the gluteus maximus and quadriceps muscles are *extensor* muscles, abnormalities of these muscles produce characteristic findings during the *stance* phase. Because the foot dorsiflexors (i.e., the weak muscles causing foot drop) are *flexor* muscles, abnormalities produce characteristic findings during the *swing* phase. In the weak gluteus maximus gait (*top row*), there is an abnormal backward lean during stance. In the weak quadriceps gait (*middle rows*), patients may hyperextend their knee during stance (i.e., genu recurvatum, *second row*) or place their ipsilateral arm on the leg to prevent the knee from buckling (*third row*). In the foot drop gait (*bottom row*), the actual foot weakness is conspicuous (*bottom arrows*), and there is excessive flexion of the hip and knee during the swing phase (*upper arrow*) and a slapping sound of the foot when it strikes the ground.

main role of the quadriceps during gait is to support the flexed knee during stance, and patients with weak quadriceps avoid bearing weight on a flexed knee by hyperextending the joint (i.e., genu recurvatum). They can fully extend the knee because their hip flexes strongly during swing and then decelerates abruptly, which whips the tibia forward.[16] Alternatively, other patients with weak quadriceps may place their hand just above the knee to support the weak leg and prevent the knee from buckling during stance (see Fig. 7.3). Most patients with weak quadriceps muscles have great difficulty walking on uneven ground.

4. FOOT DROP (WEAK TIBIALIS ANTERIOR AND TOE EXTENSOR MUSCLES)

There are two characteristic features: (1) foot slap, which is the uncontrolled slap of the forefoot immediately after the heel makes contact, thus producing (in patients with unilateral foot drop) a characteristic cadence of two sounds alternating with a single sound (i.e., stance of abnormal foot alternating with that of normal foot): "dada…da….dada….da"; and (2) steppage gait, which occurs during the forward swinging phase of the affected foot, when the patient flexes the hip and knee excessively in order to clear the foot from the ground, thus creating the appearance of the abnormal foot "stepping over" an invisible object (see Fig. 7.3).[15]

D. SPASTICITY

Spasticity is a feature of weakness of the upper motor neuron type (see Chapter 61). Characteristic gaits are the hemiplegic gait and diplegic (paraplegic) gait.

1. HEMIPLEGIC GAIT

This gait is the result of poor control of the flexor muscles during the swing phase and spasticity of the extensor muscles acting to lengthen the affected leg (compared to the healthy side). The ankle is abnormally flexed downward and inward (equinovarus deformity), and initial contact during stance is abnormal, along the lateral edge of the foot or forefoot. The knee is stiff, hyperextends during stance, and does not flex normally during swing. The contralateral step often advances just to meet the position of the paralyzed limb, instead of advancing normally beyond it.

Because the paralyzed leg is hyperextended, and therefore longer than the sound leg, the patient may drag the toe of the affected leg during swing or adopt abnormal movements to clear that limb during the swing phase. These movements include contralateral trunk lean, which raises the ipsilateral pelvis to clear the paralyzed leg,

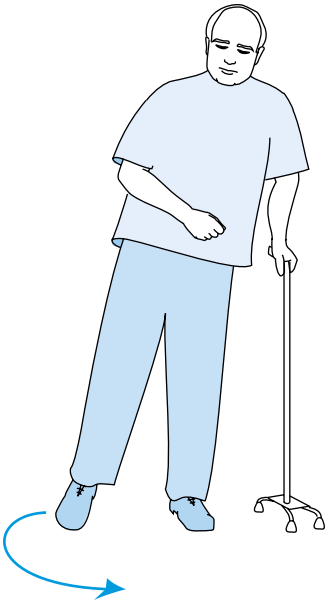


FIG. 7.4 HEMIPLEGIC GAIT. In a patient with right hemiparesis, the paretic arm is flexed and the paretic leg is hyperextended. In order to clear the extended right leg from the floor, the patient leans over the healthy left leg and slowly advances the stiffened, paralyzed right leg with a circumducting movement (*arrow*).

and circumduction, an abnormal movement in which the toe traces a semicircle on the floor, first moving outward and then inward as it advances, instead of a normal straightforward movement (Fig. 7.4).

According to classic teachings, the clinician should suspect mild hemiplegia if a patient swings his or her arms asymmetrically while walking, although this finding appears in 11% to 70% of normal persons[24,25] and the sign did not accurately detect focal cerebral disease in one study (sensitivity 22%, specificity 89%, positive and negative LRs not significant).[24]

2. DIPLEGIC GAIT

Diplegic gait affects patients with spinal cord disease (e.g., spinal cord trauma, cervical spondylosis, B12 deficiency). The combinations of spasticity and abnormal proprioception cause a characteristic slow, laborious, and stiff-legged gait. In some spastic diplegias of childhood, adductor spasm causes the feet to cross in front of each other (scissors gait).

E. RIGIDITY

Chapter 61 describes the characteristic features of rigidity and distinguishes it from spasticity. The most common gait abnormality due to rigidity is the parkinsonian gait.

1. THE PARKINSONIAN GAIT (FIG. 7.5)

The characteristic features are (1) flexed posture of the arms, hips, trunk, and neck; (2) rigidity of movement (en bloc turning, difficulty initiating gait); (3) steps that are flat-footed, small, and shuffling, with a narrow base; (4) diminished arm swing

FIG. 7.5 **PARKISONIAN GAIT.** The characteristic features are flexed posture (trunk, neck, and arms), diminished arm swing, narrow-based gait, and shuffling steps.

(normal arm excursion, measured at the wrist, averages 16 inches; the average value for patients with Parkinson disease is 5 inches); (5) involuntary hastening of gait (festination); and (6) poor postural control (retropulsion).

2. DIFFERENTIAL DIAGNOSIS

Patients with spinal stenosis superficially resemble those with Parkinson disease in that they have a flexed stance (simian stance), which reduces the tension on the lumbosacral nerves.[26] Patients with spinal stenosis, however, complain of pain and otherwise have a normal gait.

The distinguishing features of the frontal gait disorder, which also may superficially resemble the parkinsonian gait, are discussed later in the section on Frontal Gait Disorder.

3. CLINICAL SIGNIFICANCE

Patients presenting with parkinsonism (i.e., bradykinesia in combination with rigidity, resting tremor, or both) have either Parkinson disease (a disorder from pathologic depigmentation of the substantia nigra that responds to levodopa) or a group of mimicking disorders called Parkinson-plus syndromes (disorders with distinct pathologic findings that respond less well to levodopa; e.g., progressive supranuclear palsy and multiple system atrophy; see Chapter 66).

The gait of patients with Parkinson disease has a narrower base than the gait of patients with the Parkinson-plus syndromes, suggesting that Parkinson-plus patients

may have greater instability during tandem gait. In clinical studies of patients *with parkinsonism*, the ability to successfully walk 10 tandem steps without errors thus increases probability of Parkinson disease (LR = 5.4, EBM Box 7.1); inability to complete 10 tandem steps, in contrast, increases the probability of a Parkinson-plus syndrome (LR = 4.6; see Chapter 66).

F. ATAXIA

The characteristic features of the ataxic gait are its wide base and irregular, uneven, and sometimes staggering steps. (The normal base, measured when one limb swings past the other at mid-stance, is 2 to 4 inches.) There are two types of ataxia: sensory ataxia and cerebellar ataxia.

1. SENSORY ATAXIA

Sensory ataxia affects patients with significant proprioceptive loss (see Chapter 62). Characteristically, the patient looks down and walks as if throwing his feet, which tend to slap on the ground. Smooth, familiar routes cause less trouble than uneven, rough ones.

2. CEREBELLAR ATAXIA

Affected patients place their feet too far apart or too closely together irregularly, and they sway, stagger, and reel in all directions as if intoxicated by alcohol. In contrast to sensory ataxia, patients with cerebellar ataxia have other cerebellar signs, including dysmetria, hypotonia, intention tremor, dysarthria, and nystagmus (see Chapter 65).

3. ROMBERG SIGN

A. INTRODUCTION

In his famous textbook, written between 1840 and 1846, Moritz Romberg described the sign now bearing his name as a finding in patients with severe sensory ataxia from syphilitic damage to the dorsal columns of the spinal cord (tabes dorsalis). According to Romberg, when a patient with tabes dorsalis stands and closes his eyes, "he immediately begin to moves from side to side, and the oscillations soon attain such a pitch that unless supported, he falls to the ground."[30] Most authors claim that the Romberg sign is negative in patients with cerebellar ataxia, although Romberg did not make this claim (cerebellar disease was not defined during his time; Duchenne and Babinski later added this diagnostic point).[31]

B. DEFINITION OF A POSITIVE ROMBERG SIGN

One problem with the Romberg sign is that various authors define the positive test differently: some state that it is the increased swaying that occurs when the eyes close, while others require the patient to be on the verge of falling down.[30] Increased swaying alone seems inadequate, because most normal persons sway more when they close their eyes, as do patients with vestibular, cerebellar, and Parkinson disease.[32]

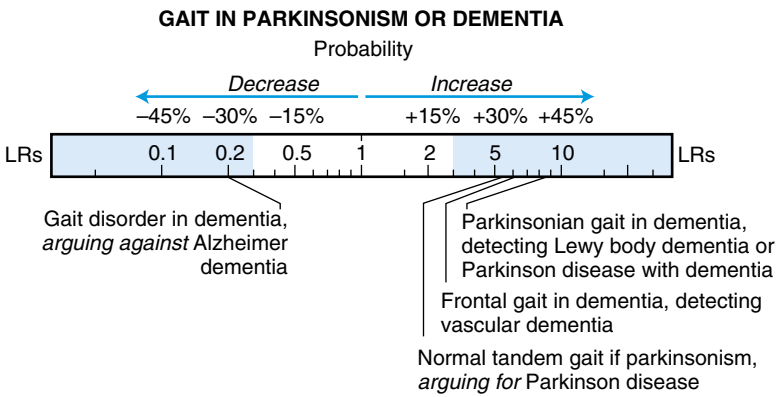
The best definition of a positive Romberg sign is an inability to stand for 60 seconds with feet together and eyes closed. In one study, every healthy person and over half of the patients with cerebellar ataxia could maintain this position for 60 seconds, whereas half of the patients with sensory ataxia lasted only 10 seconds before beginning to topple over.[33]

A related sign, the sharpened Romberg sign,[34] in which patients must stand with one foot in front of the other with eyes closed, has little proven diagnostic value. Many normal persons, especially elderly ones, are unable to stand like this for very long.[33]

EBM BOX 7.1
*Gait Abnormalities in Patients With Parkinsonism or Dementia**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is	
			Present	*Absent*
Detecting Parkinson Disease, in Patients With Parkinsonism				
Able to perform 10 perfect tandem steps[27,28]	67-92	82-91	**5.4**	**0.2**
Detecting Type of Dementia§				
Any gait or balance disorder (moderate or worse), detecting Alzheimer dementia[29]	16	25	**0.2**	**3.4**
Parkinsonian gait, detecting Lewy body dementia or Parkinson disease with dementia[29]	78	91	**8.8**	**0.2**
Frontal gait, detecting vascular dementia[29]	56	91	**6.1**	0.5

*Diagnostic standard: For *Parkinson-plus disorder*, the conventional diagnostic criteria for multiple system atrophy, progressive supranuclear palsy, Lewy body dementia, corticobasal degeneration, or vascular dementia;[27] for *Alzheimer dementia*, conventional diagnostic criteria.
†Definition of findings: For *unable to perform tandem gait,* the patient was instructed to take 10 consecutive tandem steps along a straight line without walking aids and support, with eyes open, and the clinician observed ≥1 side step during testing.[27]
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
§All patients have dementia.
Click here to access calculator



G. FRONTAL GAIT DISORDER

1. DEFINITION

Frontal gait disorder is an imprecise term describing a combination of findings seen in patients with cerebral tumors, subdural hematomas, dementing illness, normal pressure hydrocephalus, and multiple lacunar infarcts.[35,36] The characteristic findings are (1) slow, shuffling, wide-based gait (marche a petis pas); (2) hesitation in starting to walk (ignition failure); (3) difficulty picking feet off the floor (magnetic foot response); and (4) poor postural control. Motor function of the legs is sometimes much better when these patients are seated or lying down, suggesting an element of gait apraxia.

Some of these findings resemble parkinsonism, but the distinguishing features of the frontal gait disorder are its wide base, normal arm swing, absence of other parkinsonian features, more upright posture, and higher incidence of dementia and urinary incontinence.

2. CLINICAL SIGNIFICANCE

In studies of elderly patients undergoing computed tomography (CT) of the head because of neurologic symptoms, the finding of a frontal gait disorder correlates strongly with the CT finding of ventricular enlargement.[10,37,38] Only a minority of these patients, however, meet the criteria for normal pressure hydrocephalus, suggesting that the findings of ventricular enlargement and gait disturbance are general ones occurring in many different forebrain disorders.[10,37]

Analysis of gait assists diagnosis of patients with dementia. The presence of a gait disturbance makes Alzheimer disease less likely (especially if the gait disturbance appears early during the patient's course; LR =0.2, see EBM Box 7.1); a parkinsonian gait in patients with dementia increases the probability of Lewy body dementia or Parkinson disease with dementia (LR = 8.8), and a frontal gait increases the probability of vascular dementia (LR = 6.1).

IV. EVALUATION OF GAIT DISORDERS

The methods of evaluating gait range from very simple tests that require minutes to complete (e.g., assessing the fall risk in elderly patients) to comprehensive observational gait analysis, which physiatrists use to break down complicated gait abnormalities into smaller components to direct treatment.[16] Most clinicians adopt an intermediate approach and ask the patient first to walk back and forth several strides at a time, and then again on the toes, heels, and using tandem steps, all maneuvers that may bring out weak muscles or difficulties with balance.

Testing gait is essential, whatever the method, because patients often appear normal during conventional tests of motor, sensory, musculoskeletal, and visual function, yet when asked to stand and walk, demonstrate abnormal balance and gait.[39]

A. OBSERVATIONAL GAIT ANALYSIS[15,16]

Using this method, the clinician focuses on one limb at a time as the patient walks, first observing the ankle, then the knee, hip, pelvis, and trunk. At each joint, the clinician considers each of the four fundamental ingredients of abnormal gait: pain, immobile joints, muscle weakness, and abnormal limb control.

As an example, the differential diagnosis of "abnormal ipsilateral trunk lean during stance" includes ipsilateral hip pain, ipsilateral short limb (>1.5 inches shorter), or intentional attempts to clear the contralateral limb during swing (e.g., drop foot or extended limb). Also, "dragging of the foot or toe during swing" may occur because of weak ipsilateral ankle dorsiflexor muscles, ipsilateral plantar flexion contractures, inadequate ipsilateral hip or knee flexion, or impaired proprioception. An excellent manual of observational gait analysis by the Rancho Los Amigos Medical Center has been published.[15]

B. PREDICTING FALLS

Thirty percent of persons over the age of 65 living in the community fall each year.[4] Of the many brief tests designed to identify patients at higher risk for falls, the best studied are "stops walking when talking," and "timed up-and-go" tests. In studies of these tests, the history of a prior fall during the previous year predicts another fall in the next 6 to 12 months, with a sensitivity of 20% to 62%, specificity of 71% to 93%, and positive LR of = 2.4.[4,40,41]

1. THE FINDINGS

A. STOPS WALKING WHEN TALKING

The premise behind this test is that elderly patients at risk for falls have difficulty completing separate tasks simultaneously. To perform the test, the patient is accompanied while walking and then observed to determine what happens when the examiner initiates conversation. If the patient stops walking when talking, the test is positive.

B. TIMED UP-AND-GO TEST[3]

The clinician measures the time it takes the patient to rise from a standard chair, walk to a line on the floor 3 meters away, turn, return, and sit down again. They are instructed to walk at normal speed and are allowed one trial before timing. The timing starts when the patient's back comes off the chair and ends when their buttocks touch the seat of the chair.

2. CLINICAL SIGNIFICANCE

According to the LRs presented in EBM Box 7.2, the most compelling findings increasing a patient's risk of falls are failure to stand with feet together and eyes open for 10 seconds (LR = 4.5), a positive "stops walking when talking" test (LR = 3.0), a positive palmomental reflex (LR = 2.8, see Chapter 63), and a "timed-up-and-go" test of 35 seconds or more (LR = 2.6). A timed-up-and-go test result of less than 15 seconds identifies patients at low risk of falls (LR = 0.1). The cutoff points used for the timed-up-and-go test vary greatly and likely depend on methodology and specific population studied;[45] the LRs in EBM Box 7.2 are derived from study of frail nursing home residents.

V. CANES

Physical examination of gait is incomplete without considering the length of the patient's cane and which arm the patient uses to hold the cane.

A. LENGTH OF CANE

Twenty-three percent to 42% of the time, the patient's cane is too long or too short by 5 cm or more.[46,47] An appropriately fitted cane should extend the distance from the distal wrist crease to the ground when patients wear everyday shoes and dangle their arms at their sides.[48]

B. CONTRALATERAL VERSUS IPSILATERAL USE OF CANE

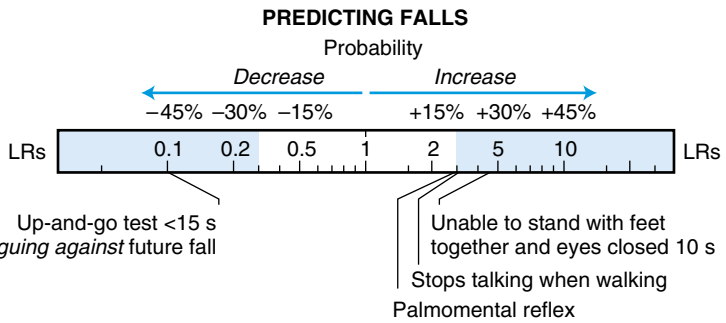
In patients with hip and knee arthritis, patients are conventionally taught to hold the cane in the contralateral hand, although compelling evidence for contralateral cane use exists only for patients with hip arthritis.[49,50] By placing just 20, 33, or 38 pounds of pressure on a cane contralateral to a diseased hip when standing on that hip, the patient can *reduce* the pressure on the diseased femoral head by 165, 272, or 319 pounds, respectively.[49]

The references for this chapter can be found on www.expertconsult.com.

EBM BOX 7.2
*Predicting Falls**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is *Present*	Likelihood Ratio‡ if Finding Is *Absent*
Neurologic Examination				
Palmomental reflex present[4]	31	89	2.8	0.8
Failure to stand with feet together and eyes open for 10 s[40]	4	99	**4.5**	NS
Failure to tandem walk (>2 errors)[40]	53	70	1.7	0.7
Special Tests				
Stops walking when talking[2,42-44]	14-53	70-97	**3.0**	NS
Timed-up-and-go test[41]				
<15 s	4	67	**0.1**	…
15-35 s	60	…	NS	…
≥35 s	36	86	2.6	…

*Diagnostic standard: For *falls*, ≥1 fall during 6-month follow-up[2,41-44] or 12-month follow-up.[4,40,42]
†Definition of findings: For *palmomental reflex*, see Chapter 63; for all other tests, see text.
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
NS, Not significant.
Click here to access calculator



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